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A CLINICAL AND PATHOLOGICAL CONTRIBUTION TO THE
STUDY OF THE CENTRAL LOCALIZATION
OF THE SENSORY TRACT.¹

BY HERMAN H. HOPPE, A.M., M.D., OF CINCINNATI, O.

The object of the present report is to throw some light upon the subject of cerebral, especially cortical localization of sensory functions.

Much good work has been done physiologically and experimentally in the last few years, especially by Dejerine, Long, Probst, von Monakow and others on the functions of the thalamus opticus, and some ideas concerning the course of the sensory tract from the thalamus to the cortex will in a measure at least have to undergo a modification.

What we will specially consider in this paper are (a) lesions of the thalamus, (b) lesions of the area supplied by the middle cerebral artery, (c) cortical and subcortical tumors of the Rolandic region, to see what light they can throw on the course and cortical termination of the sensory fibers.

Physiologically speaking, the subject of our study is the thalamus opticus and the thalamo-cortical neurone, and the cortico-thalamic neurone. It has been established as an anatomical fact, by the researches of Dejerine, Long, Probst, von Monakow, Kölliker, Bechterew, Flechsig, Schaefer, and Spiller that:—

1.—The thalamus opticus is connected by fibers with a large

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section of the cortex, and reversely that the cortex is connected with the thalamus opticus. Thus we have thalamo-cortical and cortico-thalamic neurones.

2.—That all the centripetal sensory tracts end in the thalamus. No centripetal sensory tract (Probst) ever passes through the thalamus to proceed to the cortex.

3.—That the thalamo-cortical bundles pass through all parts of the posterior limb of the internal capsule, on their way to the cortex. They are not massed in one bundle, like the pyramidal tract, but in a fan-shaped way they pass along with or through the fibers of the posterior limb of the capsule. (Dejerine and Long.)

4.—The thalamo-cortical neurones spreading out in a fan-shaped way pass to the cortex of the frontal, parietal, occipital and temporal lobes.

The fibers from the lateral and ventral nuclei, especially, pass to the central convolutions around the fissure of Rolando, the gyrus fornicatus and the posterior portions of the frontal lobes. These observations of Probst on lower animals have been verified by Flechsig in the fetal human brain. The physiological results obtained are far from being as exact as the anatomical and histological. The question involved is; what relation does the thalamo-cortical neurone bear to the conduction and perception of sensory impressions, coming from skin, muscles and ligaments.

Three anatomical points are involved in these neurones (a) the thalamus opticus, (b) the cortex, (c) the fibers connecting the thalamus with the cortex.

Wundt found that the cutting in two of the thalamus opticus was followed by a change in movement of the animal in such a way that if it wished to go forward in a straight line, it invariably made a circular movement.

Nothnagel in his experiments of producing small areas of destruction in the thalamus was unable to find that any disturbance of sensation followed such lesions. He also produced bilateral lesions of the thalamus, and the animal showed no effects whatever from the lesions—all movements were normal; no paralysis, no disturbance of skin sensation.

Ferrier found that destruction of the thalamus was followed by sensory disturbance of the opposite side of the body. He at-

tributes only sensory functions to the thalamus, and holds that it is devoid entirely of motor functions.

Meynert says that the thalamus is an organ for complex or combined movements, which are executed unconsciously as reflex movements, resulting from the sensory impressions brought to the thalamus from the sensory end organs. These thalamus perceptions are brought to the cortex by way of the thalamo-cortical fibers and are treasured there as memory pictures, and become the means or basis of future voluntary movements. (Nothnagel agrees to this.) The only positive and constant sensory result of an injury to the thalamus, as proven by von Monakow, is the hemianopsia, and this occurs as a result of a destruction of the pulvinar.

Edinger saw intense pain in the opposite extremities in a lesion of the thalamus. Others saw tremor.

Von Monakow thinks that a part of the reflex sensory arc is located in the thalamus. His idea is as follows:

The thalamus opticus is an important link in the central apparatus for the perception of impressions made upon the special senses and the skin, more especially on the latter. The fact that lesions resulting from destruction of the thalamus in whole or in part, are often only temporary or even absent altogether is due to two factors; first, that the senses including general sensations have a bilateral representation (?) and secondly, that the sensory impressions may follow vicarious routes, if those which they usually follow happen to be destroyed.

The great differences in results obtained by different experimentors, physiologically, may be due to a faulty technique, as Probst says, and the destruction of surrounding areas of the cerebrum. Probst has done much careful work on the functions of the thalamus. He divides the lesions into two groups; (A) passing, (B) permanent.

(A) Some clumsiness in the use of and placing of the opposite foreleg.

(B) (1) Circle movement. (2) Abnormal position and holding of head. (3) Hemianopsia.

Sensation on opposite side seemed at first diminished, afterward this condition was doubtful.

He lays special stress upon the fact that there is never any

permanent or absolute loss of sensation in dogs after thalamus injuries. There is no disturbance of reflexes, no loss of motor power, no galoppe movements such as were described by Fournie.

Probst also lays great stress upon the fact that previous experiments on animals were not followed by careful anatomical serial sections of the brain, and were therefore defective.

When we read, however, of the experiments of Nothnagel and of Ferrier, who are both capable experimentors and careful observers, and whose experiments lead to exactly opposite conclusions, so far as sensation is concerned, we are at a loss to account for the difference, except perhaps that the sensory tracts and centers are different for cats, dogs and monkeys.

The reason which Probst advances for the different results obtained may hold good for such instances where positive results were obtained, but would hardly hold good for negative results, at least as far as sensation is concerned, such as Nothnagel obtained.

The clinical investigations in man are also contradictory as far as the thalamus is concerned. Some writers attribute vision functions to the thalamus, others deny this flatly; some look upon the thalamus as a great sensory center, others deny this just as vehemently; some deduce motor paralysis from lesions of the thalamus, others deny that they occur.

Sancerthe and Foville, Lussana and Lemoique say that the thalamus presides over movements in the upper extremities. Cohn, Gintrac, Nothnagel, Bastian, hold that if the lesion is strictly limited to the thalamus, motor signs are not seen.

Todd, Carpenter, Broadbent, attribute sensory functions to the thalamus. This is denied by Vulpian and Lafforque. Long says that thalamic lesions produce permanent hemianesthesia. Chrichten Brown holds that lesions of the thalamus not only destroy sensations, but also reflex irritability for the opposite side of body. This is denied by Moeli and Lafforque.

Nothnagel says if, as a result of a focal lesion, we have a hemiplegia with involvement of the face, and if the face muscles cannot be moved by the will, but if emotionally both sides of the face are moved equally, we can say that the thalamus and the cortical connections are intact.

Disturbance of vision after lesions of the thalamus are indefinite.

Lesions of the posterior part of the thalamus are associated with rapid muscular atrophy of the hemiplegic side (Borgherini, Eisenlehr, Darkschewitsch, Quinke). Eisenlehr and Von Monakow deny that this always occurs.

Gross lesions of the thalamus may lead to mistakes because of the fact that other structures may be involved, as, for instance, in hemorrhages or tumor. The cases may present more signs and symptoms than those due solely to a disturbed function of the thalamus. On the other hand, if such cases do or do not present signs which prove the present theory of the functions of the thalamus, even though there be additional signs and symptoms caused by the destruction of tracts whose functions we know, they may prove of physiological value. Therefore, it is strange to relate that Probst himself should lay but little stress on a case in which sensory manifestations during life were negative, but the autopsy showed a destruction of both thalami by a tumor. The fact that this growth was very extensive is not of any importance compared to the fact that both thalami optici were destroyed without producing sensory manifestations. On the other hand, he publishes a case of hemorrhage limited to the thalamus and internal capsule in which during life there were hemiplegia, hemianopsia, hemianesthesia and nystagmus. He found in the autopsy a complete destruction of the thalamus with secondary degeneration of the thalamo-cortical fibers. He attributes the hemianesthesia, the nystagmus and the hemianopsia, together with the spastic condition of the muscles, to the destruction of the thalamus.

Case I.—B. W. F. male, aged 70 years. Admitted March 16, 1899, with history of feeling badly for two or three days, complained of occipital headache. On the night of March 16, while walking along the street, suddenly fell and became unconscious.

Examination: Complete motor paralysis of the right side of face, right arm, partial motor paralysis of the right leg.

Sensation: Right side, pain; shows that he feels pin-stick on side of face by facial expression, and also of left arm by movement of arm; heat and cold, negative.

Post-mortem twelve hours after death: Brain arteries show marked atheromatous and calcareous changes, left lateral ven-

tricle full of clotted blood, softening and degeneration of internal capsule, lenticular and caudate nuclei and optic thalamus softened, brain diffuent, calcareous arteries plainly seen in softened mass.

While the mental condition of this patient would not allow of a careful examination of sensation in all of its qualities, still it is positive that the perception of pain was present in the paralyzed extremities, with an extensive softening, which included the thalamus opticus.

Case II.—Tumor of brain, paralysis and aphasia. M. B. Female, aged thirty-two years, married. Admitted to City Hospital February 8, 1899, with history of blindness which began eight months ago, paralysis six months ago. On admission semi-comatose; very stupid; talks slowly when questioned; volunteers no statements; sleeps most of the time; hard to arouse when asleep; can give no account of herself.

Examination: Right arm completely paralyzed, right leg partially so; left arm seems very weak, left leg also weak.

Sensation: Pain diminished on right side; heat, cold and touch negative on account of mental condition. Died April 7, 1899.

Post-mortem twelve hours after death: Brain, calvarium removed; skull opened revealed a tumor mass involving left lobe, probably sarcomatous in character; very vascular; apparently began in the optic thalamus and involves ventricles and almost entire left lobe of brain; areas of softening in the lower surface of temporal lobe, both sides; degenerative changes in the optic chiasm.

In this case of extensive brain tumor of the left hemisphere, causing destruction of the thalamus, we find a diminution but not complete loss of pain sense.

Of all the numerous cases of tumor and softening of the thalamus on record, very few show a complete hemianesthesia, many a slight diminution of sensation, but by far the majority of all cases expressly say that sensation was normal. Up to five years ago, Oppenheim gives rather a complete digest of the sensory symptoms in published cases, and since this there have appeared the cases of Fraenkel, Bary, Beevor, Schule, Jacobson, Luxembourg, Bayerthal, Probst, Boost, and Courtney, in which complete destruction of the thalamus—and, in Jacobson's case, of both thalami—was followed by no disturbance of sensation.

Schaffer's case is of great interest in that it offers positive evidence of the function of the white connecting fibers between the thalamus opticus and the cortex. Gorden's observations con-

cerning sensory lesions in motor hemiplegia are clinical and have no pathological weight.

Schaffer holds that hemianesthesia is produced by lesions which destroy the connection between the thalamus and cortex of the Rolandic area. He holds that we can no longer maintain Charcot's theory of a separate sensory tract, but that the fibers of the latter are mingled with those of the pyramidal tract. He bases his opinions on a very careful autopsy made upon a case which during life presented a complete hemiplegia and hemianesthesia, a softened area was found occupying the head and tail of nucl. caudatus, anterior half and knee of internal capsule, cortex along the horizontal limb of fissure of Sylvius. There was no involvement of the thalamus, but the microscopic examination showed a degeneration of nucleus lateralis and of the thalamus. The posterior half of the internal capsule was free. He concludes that since the area destroyed by the softening is usually occupied by the pyramidal tract and that clinically the symptoms were complete hemianesthesia and hemiplegia, the sensory and motor tracts are identical.

It does not seem to me that this case proves any more than the existence of a cortico-thalamic neurone, the destruction of which may cause a permanent sensory paralysis. It does not prove that the fibers destroyed were all connected with the Rolandic area, many of the fibers destroyed may have been those which go to the parietal lobe.

Let me introduce at this point two cases of extensive subcortical tumors of the Rolandic area.

Case III.—Subcortical tumor below right arm center, occupying centrum semiovale and nucleus lentiformis. No disturbance of sensation, complete paralysis and no hemianesthesia. The paralysis in this case was due to the destruction of the internal capsule and corona radiata.

R. G., aged ten, colored, was admitted to Cincinnati Hospital. The family and personal history were negative. There had been a gradual development of weakness and stiffness of the left arm and leg. This had lately increased to such an extent as to interfere with the use of the left arm altogether. The left leg was stiff and rigid, and was dragged during walking; he had no pains in the extremity.

Examination gave the following: Mental condition was dull

and apathetic; there was a tendency to somnolence; when aroused the mind was clear. Pupils were equal in size and reacted to light; papilla was swollen and injected; there was choked disc; the external muscles of the eyes were normal in function. There was no defect in sensation of face or scalp; the left side of face, lower half, was paretic; the tongue protruded to the left. The left arm was rigid and spastic; the radial and triceps reflexes were increased; the muscular strength was reduced almost to a minimum; the size, the surface temperature and the sensation of arm were normal. The leg was normal in size and temperature, the muscles rigid and hard, the limb in condition of spasticity, the reflexes increased. There was ankle clonus; muscular power was almost entirely lost; he was scarcely able to walk; dragged the leg; the sensation was normal. The diagnosis was spastic hemiplegia, subcortical brain tumor.

The skull over this region was trephined, and a piece of bone two inches in diameter was removed; the bulging of the brain and membranes was very marked; but on opening the dura the cortex of the brain was found intact. An incision was made through the cortex about an inch in length; but, as proved later, this was not deep enough. No tumor being found, the wound was closed. Death occurred six hours later from shock. The autopsy showed a soft yellowish mass, fully the size of a hen's egg, occupying the region of the nucleus lentiformis of the right side; it extended outward to within one-half inch of the cortex, extending upward into the region of the ascending frontal convolution and parietal lobe, viz.: the centrum semiovale; inward and downward it extended to the internal capsule. The microscopic examination showed the growth to be a tubercle.

Case IV.—Tumor, cortical and subcortical of the foot center. Jacksonian epilepsy. First seven years Jacksonian epilepsy was not followed by paresis, but only by subjective sense of numbness. Gradual development of hemiplegia. Objective sensation normal.

J. H. was admitted to Cincinnati Hospital. He complained of numbness of the left arm and leg, followed by affected areas, lasting about half an hour. His family history was negative.

About seven years before, while ascending a ladder, his left leg suddenly became helpless, causing him to make a misstep, and he fell a distance of fifteen feet, but as he fell on a sofa he sustained no injury at the time. He did not lose consciousness, but immediately felt numb in the entire left half of the body; this feeling lasted about twenty minutes. After the numbness passed away he went to work, feeling no ill effects. About a month later he was seized in the same manner, and the attack lasted about the same length of time. His paroxysms during the past years may be summed up as follows: (1) Frontal headache, last-

ing about twenty minutes; (2) vomiting, the vomitus consisting principally of bile; (3) tonic spasms of left arm and left leg; (4) numbness and weakness, lasting about thirty minutes, after an attack.

Examination: His cerebation was somewhat slow, and he had a slight difficulty in talking.

Optic neuritis; pupils react to light and accommodation; third, fourth and sixth nerves were apparently normal; fifth, motor and sensory normal; seventh, weakness of muscles of left side of face; ninth, tongue deviates to the left side; tenth, some difficulty in breathing in morning; eleventh, on turning head to left severe pain in right occipital region; similar pain in left on turning head to right; twelfth, normal. There was some difficulty in swallowing. Motility, muscular power and grasp of right arm apparently normal and equal. Flexion, extension and power of right leg normal. Right leg was affected at times, but only temporarily; often had passing weakness, rigidity and ankle clonus on right side. Unable to lift left leg from bed when extended, and when it is elevated cannot flex nor extend it; resists bending somewhat, but no pain on bending. Left arm becomes exhausted easily; some incoordination; slightly weaker than right.

Gait: Dragged left leg and foot; left arm hung almost motionless at side, although he had use of it; head inclined to the left side.

Sensation: Pain, tactile, heat and cold, normal.

Reflexes: Right and left knee-jerks exaggerated; right and left ankle clonus marked; right and left wrist-jerk marked; right and left elbow-jerk marked.

Exploratory trephining was done; patient died of shock.

Autopsy: The longitudinal sinus was obliterated by a soft, semi-fluid, cream-colored growth. Under the dura mater was a growth about the size of a goose egg, which began on the surface about two inches from the tip of the first frontal convolution and extended backward along the longitudinal fissure to about two inches to the fissure of Rolando.

The cortical size of this growth was about three and one-half inches, and occupied the posterior half of the first and second frontal and ascending parietal convolutions around the fissure of Rolando and backward into the gyrus angularis. It bulged into the longitudinal fissure and pressed on the median surface of the psychomotor area of the left side.

Subcortically it extended into the corona radiata, pressed on and destroyed portion of the lenticular nucleus and the fibers of the internal capsule. The cortex above the tumor was entirely obliterated, whereas the cortex of the lower portion of the psychomotor area and the portion of the corona radiata beneath it were intact.

The tumor was soft and fluctuating. On section it was semi-solid, very soft, creamy in color and rather vascular. After being hardened it proved to be non-infiltrating, and could be shelled out with ease. Microscopic examination proved the tumor to be a gliosarcoma.

Both of these cases are almost identical with Schaffer's case. In both we have a complete spastic hemiplegia. In both the corona radiata of the motor area was extensively destroyed; still there was no hemianesthesia.

Case V.—Circumscribed tubercular abscess of the lower third ascending frontal convolution, left side. Diminution of pain sense of right facial region.

F. E., male; aged forty-two years; widower; conductor. Admitted to Cincinnati Hospital March 4, 1901, in state of syncope, and no history obtainable, except has had headache for some time and has been dizzy.

Examination: Paresis of right side of face. Paresis of right arm and leg. Diminution of pain sense in face.

Sensation otherwise normal.

Died March 10, 1901.

Post-mortem: Brain when taken out was very soft and flabby, the left lobe being especially so. On the cortical surface, in an area extending downwards from the lower half of the anterior ascending frontal convolution to the base of the brain, and bounded posteriorly by the fissure of Rolando, inferiorly by the fissure of Sylvius, and anteriorly infringing on left inferior frontal and middle frontal convolutions, was an area of softening. After hardening in formalin, this proved to be a globular mass which could be shelled from its surroundings. The mass was hollow, and filled with greenish pus. Brain tissue surrounding the mass was also extensively softened. Whether this is a broken-down tumor or a tubercular abscess microscopic examination must determine.

Histological examination: Brain. From what was a cyst or cavity filled with purulent material, sections were made and stained according to two methods:

(a) Mallory's, to determine the presence of any overgrowth of glia tissue; this the specimens examined did not show. (b) Hematoxylin-eosin. The wall of the cyst, which was about $1/32$ inch in thickness, shows a very intense small-celled infiltration, with an inner layer; next the cavity ragged and in many places broken down and degenerated; while no typical tubercles were found, there were many rather small, suspicious nodular collections of cells, which were taken to represent tubercular areas; the outermost area showed a layer of thickening, which we think was glia proliferation, in spite of the fact that the Mallory specimens

were negative. There was also meningeal thickening over the area.

The location of this tubercular abscess, secondary to pulmonary tuberculosis, explains the aphasia, and also the facial paresis, as it involved the lower half of the frontal region on the left side (speech center) and extended back to the precentral convolution; the arms and leg centers were not included in its territory. No study of secondary degeneration was made, as the brain was in a state of decomposition when obtained.

In this case we have a localized softening and tubercular abscess in the lower third of the left ascending frontal convolution, with distinct diminution of sensation in the right side of face.

The tendency has become more and more manifest in the past decade to overthrow some of our fixed ideas as to the motor areas of Hitzig and Fritsch.

H. Munk says: "After total extirpation of arm and leg area, tactile sensibility of opposite extremities is permanently lost. This conclusion, verified apparently by Schiff, is denied by F. A. Shafer in a paper read by him before the Congress of Physiologists at Cambridge, England, August, 1898. *J. Physiol.*, Vol. xxxii., p. 310. Shafer operated on thirty monkeys.

His conclusions are : (a) A complete voluntary motor paralysis of a part may be produced by cortical lesion without perceptible loss of tactile sensibility. (b) Motor paralysis, which is produced by a lesion of the Rolandic area, is not the result of sensory disturbance. (c) Tactile sensibility is not localized in the same part of the cortex from which voluntary motor impulses directly emanate. He says that it is certain that the motor cortex receives afferent sensory fibers from the sensory region of the brain; that the irritation or destruction of these fibres explains the subjective and slight objective disturbances seen in motor paralysis.

Notwithstanding these observations, Munk persistently refuses to call the Rolandic area by any other name than the body-sensation area, and his opinion has gradually gained ground, backed by such authorities as Flechsig, Dejerine, Luciani, Sepilli, who hold the extreme view that the sensory and motor areas are identical. These views have always been opposed with much vigor by Hitzig, who claims that his original views concerning the motor functions of the Rolandic area have full value to-day.

Ferrier holds that the sensory areas are in the temporo-sphenoidal lobe.

Schaffer has recalled his views concerning the gyr. fornicatus.

Horsley held that the gyr. fornicatus is the sensory center, but has modified his views to the extent of attributing tactile and locomotion sense to the motor area.

Flechsig limits his "body sensory" sphere to the ascending frontal and parietal convolutions, a slight part of the frontal lobe, the supramarginal gyrus and the gyr. subangularis. He holds that motor and sensory cortical areas are located side by side in this region. He denies any sensory functions whatever to the other parts of the parietal lobe.

Von Monakow, whose views are shared by Starr, Dana, Openheim (Tenth Int. Med. Cong., Paris); *Neurologie*, page 105), says, "that pathological investigations prove that these centers are more extensive than the embryological researches of Flechsig would seem to indicate." "There appears to exist a fundamental difference in manner in which the motor and sensory fibers radiate. The motor bundles issue from the point of reunion (pyramidal tract in the internal capsule), sharply defined, distributed in and around the motor zone, while the sensory centripetal fibers spread themselves out in the cortical sphere over a large surface, not sharply defined, in such a way that in the cross section of the radiation the central portion shows the fibers more densely packed. The sensory fibers and their centers in the thalamus degenerate only to a slight degree, and only when almost the entire cortical connection is destroyed. Their degeneration is not at all in direct proportion to the primary lesion, as is the case with the pyramidal tract. The method of secondary degeneration is therefore not of much avail in the laboratory study of cortical localization of sensory lesions, and we must fall back upon clinical and pathologico-anatomical investigations."

Case VIII.—Weakness of both arms and legs; complete aphasia. Forced turning of head and neck to left. Left-sided hemianalgesia and thermanesthesia. Bilateral thrombosis of middle cerebral arteries. Old atrophy of right lower third of Rolandic area. Acute cortical softening of the left island of Reil, post. ascending parietal, the anterior portion of first and second parietal convolutions, and the first temporo-sphenoidal convolution.

History: W. T. aet. 56; admitted to "L" Neurol. February 27, 1902.

Previous history: Had repeated paralytic strokes during past six months. Had one on the night before admission.

Present history: Admitted to ward late at night; unable to speak; apparently right side of body paralyzed; next morning arose; was found sitting on chair, dressed; unable to talk; continually holds head in position toward left shoulder.

Examination: Mental condition apathetic; unable to speak; is not comatose nor semi-comatose; pupils equal in size and react to light. External muscles of eyes apparently normal in function; eyes are not turned to the left. No nystagmus; right angle of mouth lower than the left; tongue normal.

Sensation of face: Perhaps a slight diminution for pain and temperature, but not positive.

Paresis of both arms, but can move and use both to some extent.

Paresis of both legs, ankle clonus of the right foot; no exaggeration of patellar reflexes; foot reflex of the right foot diminished; flexion of toe (no Babinski); foot reflex of left leg enormously exaggerated; apparently that of arms also.

On third day had a new apoplectic attack *without loss* of consciousness. This attack was followed by:

(a) Complete motor paralysis of right side, lower half of face, arm and leg.

(b) Complete analgesia of right side of face, arm and leg and trunk.

(c) Complete thermanesthesia for right side, face, arm, trunk, and leg.

(d) Reduction of all reflexes and total abolition of foot reflex.

On account of complete aphasia, an examination of sense of touch, muscle sense, spacing sense, and stereognostic sense was impossible. After last attack, reaction of left pupil to light absent.

Pulse, 144; small, easily compressible. Without undergoing any change from above condition, patient died seventy-two hours after the last apoplectic attack.

Post-mortem: Brain—Circle of Willis shows marked arteriosclerosis. Complete thrombosis of middle cerebral artery of the left side. Pia removed with great difficulty over posterior portion of second frontal convolution, left side, its removal showing acute softening of the posterior portion, which extends through entire gray matter into the white substance below. Lower portion of the ascending frontal convolution shows an acute softening of the gray matter, extending into the white substance. Middle and upper third of ascending frontal convolution and paracentral convolution are normal. Ascending parietal convolution, middle

third, shows acute softening. There is acute softening of the anterior part of the superior parietal convolution, with the exception of the portion adjacent to the longitudinal fissure. There is also acute cortical softening of the lower parietal convolution, of the convolution of the island of Reil, and the temporo-sphenoidal convolution, and the adjacent portion of the gyrus angularis. The beginning of the right middle cerebral artery is also thrombosed. There is an old cicatricial atrophy of the lower portion and middle third of the Rolandic area on the right side, and especially of the ascending parietal convolution; but the cortex of the entire parietal lobe and sphenotemporal lobe, with the exception of above-mentioned portions, is normal. The peduncles, pons, medulla oblongata, and the upper portion of the cord showed absolutely no *gross involvement*.

The special interest of this case, aside from the unusual condition of bilateral thrombosis of the middle cerebral artery, lies in the fact that it throws some light on the question of cerebral cortical localization for sensory impressions.

Let us first take up clinically the left side of the body—weakness of both arm and leg, enormous exaggeration of foot reflex, no exaggeration of other reflexes, no contracture. Pain sense and temperature sense normal. No statement can be made of other qualities of sensation on account of mental condition. Pathological state of right hemisphere: Old thrombosis of the middle cerebral artery, with great atrophy of the lower Rolandic area, especially the lower two-thirds of the ascending parietal convolution. This atrophy amounted almost to a disappearance of the convolution named. The remaining portions of Rolandic area, the cortex of the sphenotemporal and parietal lobes are perfectly normal. Here we have an atrophic condition of the Rolandic area, followed by paresis of the opposite side of the body, with normal parietal lobes, no loss of sensation, at least for pain and temperature.

On the right side of the body we have a complete facial and arm paralysis, almost complete leg paralysis with ankle clonus, and a complete loss of sensation, pain and temperature, for face, arm, trunk and leg.

Pathologically we find a recent occlusion of the left middle cerebral artery and an acute cortical softening, which is very marked in the anterior portion of the parietal lobe, the island of

Reil, the first spheeno-temporal convolution, and the gyr. angularis, besides affecting the posterior second frontal and a part (small) of the ascending parietal convolution.

This case proves very much concerning the cortical localization of sensory functions. (a) It proves that the psycho-motor area does not receive cortical impressions for pain and temperature, for on the left side of the brain these centers were acutely affected, as evidenced by the complete paralysis of arm and leg; and on the right side of the brain these centers, in part at least, were completely atrophic as a result of an old thrombus in the middle cerebral artery, and still temperature sense as well as the sense of pain, was normal in face and in the arm of the left side of the body. It seems to me that this case is unique in so far as both sides of the Rolandic area were affected by the same cortical process. (b) This case would seem to prove that the loss of temperature and pain sense was due to an acute cortical softening of the anterior part of the superior and inferior parietal convolutions. I take it that the softening of the island of Reil and the first temporo-sphenoidal convolution were the cause of the aphasia. Whether the mental disturbance was due to an acute process, or whether it was the result of the general brain atrophy, which was undoubtedly present, is difficult to say. The brain weighed but 1,000 gms.

This case would also prove that the sensory functions have a distinct cortical representation, for the subcortical softening was present only in spots beneath the parietal lobe, the largest of these being about the size of a franc piece and not extending more than $\frac{1}{4}$ inch beneath the gray matter. The internal capsule, nucleus caudatus, nucleus lentiformis, and the thalamus opticus, as well as the structures of the pons and medulla, were intact.

More detailed microscopic examination of this case will be made and published later by Dr. Wolfstein, Neuropathologist to the Cincinnati Hospital.

Case VI.—Subcortical tumor of the left arm center, Jacksonian epilepsy, paresis, subjective numbness after operation, soul anesthesia.

Edw. O'B., aet. thirty-one, single; no venereal disease. Onset of trouble one year ago, with attack of Jacksonian epilepsy.

The attack always preceded by numbness; numbness occurs at times without epileptic attack, in fingers of right hand.

After one year, during which he had fifteen attacks of Jacksonian epilepsy, he developed weakness of right arm and leg, with tendency to ankle clonus; especially paresis of extensor pollicis. No objective disturbance of sensation.

No choked disc, no headache; dizziness, vertigo. Left Rolandic area, tumor was found subcortical, arm center, ascending frontal convolution, and was removed.

Developed a marked hemiplegia, and after operation Dr. Zenner discovered that with a normal touch, pain and temperature sense, there was imperfect spacing sense, marked astereognosis and loss of posture sense in the fingers.

VII.—B. H., aged twenty-six years; machinist. Admitted August 6, 1898, with the following history: Last September, while at work in a coal mine, hand began to cramp; then the arm began to jerk (flexion and extension); then heart began to beat fast; then head got light, and he stood up until it made him fall; down for two or three minutes; got up and went to work. Three months later had another attack; one month later another attack. Then every three weeks. His left eye twitches when he has the spells. Had last attack three days ago. Jacksonian attacks continued almost daily.

The paresis, always present after an attack, gradually became more and more marked in the interval, until toward end of life there was present a marked paresis, more marked in the left leg than in the arm. Spastic in character, with exaggerated reflexes, ankle clonus and some contracture.

Sensation: Tactile, pain and cold normal. Heat: Seems to be some inability to recognize heat easily over inner side of left ankle and sole of left foot. Died February 3, 1899.

Post-mortem ten hours after death: Brain—Large cortical gumma not involving the dura mater; localized in the upper end of the fissure of Rolando; about $2\frac{1}{2}$ inches in diameter. The gumma was flat, occupying the gray matter, at no place more than $\frac{1}{4}$ inch in thickness, scarcely extending into the subcortical tissue and separated from the latter by a thin layer of dense, almost cartilaginous connective tissue.

Case IX.—Anna M. aet. thirty. Admitted to "N" Neurolog., October 19, 1901. Nine months ago spasm of right arm, followed by numbness and weakness. Weakness disappeared; numbness has remained. Has a continual sense of numbness, formication and pain in right hand. (This subjective disturbance of sensation was constant for two years before final operation.) Epileptic seizures occurred about once a week. Lately has had slight weakness of right arm.

Examination: Mental condition absolutely normal. Diminution of acuity of vision, choked disc, slight paresis of right side of tongue and arm; right leg not affected.

Sensation: Subjective—Pain and numbness in the right hand and forearm.

Operated upon January 8, 1903. Died.

Autopsy: Glioma, $1\frac{1}{2}$ inches in diameter, perhaps $\frac{3}{4}$ inch thick, was found occupying the middle third of the ascending frontal convolution, extending anteriorly over the frontal lobe, but not extending behind the fissure of Rolando. It is extra-cortical, but has depressed and flattened the cortex beneath it, forming a circular cavity, perhaps $\frac{3}{4}$ inch deep; the gray matter of the cortex is depressed, but not destroyed.

In both of the foregoing cases of cortical brain tumors occupying a rather extensive area and producing Jacksonian epilepsy, with monoplegia and hemiplegia, we have little or no objective disturbance of sensation, although the subjective disturbance was well marked.

If we view Case No. 6 critically, we shall see that Brissaud's views are also no longer tenable. Brissaud put forward as a hypothesis that the sensory centers were in the motor area, and that each side of the brain received impulses from both sides of the body, and that this accounted for the disappearance of sensory symptoms after the acute stage of cortical lesion. He held that cortical lesions, in order to produce permanent disturbances of sensation in the motor region, must penetrate the white substance of the subcortical region. That this cannot be looked upon as an anatomical or physiological law is abundantly proved by the above and other published cases.

Case No. 6 would tend to strengthen the contention of Knapp, Allen Starr and McCosh. From two operated cases they deduce the conclusions that tactile and muscle sense are located in the ascending parietal and other motor zones, temperature and pain sense in the parietal lobe.

Case X.—M. B., act. forty; single; mechanic. Had syphilis twenty years ago; never been nervous or hysterical. Three days ago, while seated in a chair, had sudden attack of faintness and vertigo; was afraid to rise for fear of falling; felt a sense of numbness and loss of strength in the entire left side. On attempting to use arm and leg, found that he could not do so.

Examination: Mental condition good. No change in eyes, face or tongue. Slight weakness of left arm, not marked; no change in left leg. Special senses normal. Complete thermanesthesia of left arm, left side of trunk and left leg. No increase in reflexes. Gradually improved under antisyphilitic treatment.

Two years later examination showed normal muscular power, some thermanesthesia present over left arm, left side of trunk and left thigh. In my opinion, this case was an organic and not a hysterical case.

That temperature sense must have a different localization from the other senses seems to be plausible from Case No. 10, in which, after a slight apoplectic attack, there was only to be found objectively a loss of temperature sense. More accurate localization of the special qualities of sensation have not as yet been determined, except Dana's view of the parietal lobe being specially connected with muscle sense.

One thing seems certain about the cortical sensory functions, and that is, that they are radically different from the motor functions. Destruction of small cortical areas invariably produces motor irritation, paresis or paralysis; whereas, while cortical lesions may, and at times do, produce sensory symptoms, even large and destructive lesions of the cortex and subcortical regions, such as illustrated by the Erb brain tumor case and by cases I. and II., do not produce objective disturbances of sensation.

The conclusions which we can draw are as follows:

1. Acute softening of the cortex of the area supplied by the middle cerebral artery produces, in addition to a complete hemiplegia, a complete hemianesthesia.

2. Chronic degeneration and atrophy of the lower Rolandic area, produced likewise by thrombosis of a branch of the middle cerebral artery to a very marked degree, was not followed by a permanent loss of sensation of the opposite side of face and arm.

3. Tumors destroying the cortex and subcortical regions of the motor area very frequently have subjective disturbances of sensation, such as pain, formication and feeling of numbness. Objective disturbances of sensation may occur, but are usually slight; and even in lesions producing great motor destruction, leading to complete hemiplegia, we do not usually find sensory disturbances which are at all proportionate to the motor disturbances.

In view of the fact that in the majority of cases of lesions of the cortical motor area or pyramidal tract, according to some authorities in 66 per cent. of the cases, sensory symptoms are absent; in those cases in which they are present the sensory manifestations are slight and out of all proportion to the motor symptoms, we must conclude that the sensory tracts and the cortical sensory centers are not identical with the tract or motor areas, that the extreme views of Munk, Flechsig, Dejerine, Long and others cannot be valid for man. We would rather incline to the views of Von Monakow, viz.: that general body sensations have a bilateral representation, and that sensory impressions may reach the cortex by vicarious routes, if the paths which they usually follow happen to be destroyed.

My personal view is that the chief cortical sensory areas, at least for pain and temperature, are located in the anterior portions of the parietal lobe, and that tactile and muscle sense are located in the Rolandic area.

If we sum up our experience with regard to the thalamo-cortical neurone, we know that acute hemorrhagic destruction of the thalamus opticus (Probst case), or acute hemorrhagic destruction of the corona radiata between thalamus and cortex (Schaffer case), or acute cortical softening, such as is produced by thrombosis of the middle cerebral artery, lead to hemianesthesia, and it would seem that the physiological character of the thalamo-cortical neurone as a sensory conduction tract would have to be looked upon as a fixed fact.

On the other hand, we know that chronic lesions, such as tumors and softening of either thalamus or of the subcortical white substance or of the cortex, may, and very often do, exist without causing any pronounced disturbances of sensation, although they are located in the same areas and destroy the same tissues as the acute lesions.

The acute lesions certainly do verify the newer anatomical and physiological investigations concerning the thalamo-cortical neurones. The chronic lesions leave us but one explanation, viz.: von Monakow's theory of bilateral representation and vicarious routes and centers for sensory impressions.

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